IS THE MELTZER-LYON TECHNIC FOR THE DIAGNOSIS AND TREATMENT OF GALL BLADDER DISEASE AN ASSET OR A LIABILITY?*

By WALTER C. ALVAREZ

PART I.

In 1917 Meltzer suggested, first, that inasmuch as the sphincter at the end of the common duct presumably must relax when the gall bladder contracts, if the contents of the gall bladder are to be extruded into the duodenum, the activities of the sphincter and the bladder must be coordinated and under the control of the "Law of Contrary Innervation." Second, if this be true, magnesium sulphate, when it relaxes the sphincter, must theoretically cause a reflex contraction of the gall bladder. Third, if magnesium sulphate were to be injected into the duodenum, it might help in overcoming jaundice and biliary colic.

The third suggestion was immediately put to the test by Vincent Lyon of Philadelphia. He observed that when the duodenal tube is first passed into the bowel, little or no bile can be withdrawn. Shortly after the injection of the magnesium sulphate, yellow bile appears; and shortly after that, there appears a darker and sometimes thicker bile. He assumed that the darker bile must come from the gall bladder, and the next step was to diagnose cholecystitis by the finding in this bile of pus cells, crystalline material and bacteria. Another step was to try to cure cholecystitis by repeated "drainages."

Certainly if Lyon is right in his contention this new method of diagnosis and treatment will be a great asset to the medical profession. If, however, the method is not based on a solid foundation; if it will lead to the making of wrong diagnoses and the performance of unnecessaray operations; if it will cause physicians to waste their time and their patients' time and money; and if it will encourage patients to go on with futile medical treatment until old age, starvation, myocarditis and other complications bar the way to surgical relief, it will be a liability. As I have seen of late a number of cases in which the use of the method was ill-advised and its results unfortunate, it seems to me that we must not drift along using "biliary drainage" simply because it is being done, but as scientific physicians we must stop for a few minutes to see how much grounding it has in physiologic fact, and how much likelihood there is of its ever being able to clear up the diseased conditions which we find in cholecystitis.

A review of the large literature which has already grown up about the subject shows that, for the most part, the clinical reports are very favorable. That is to be expected with any new form of treatment. On the other hand, nearly all the reports on work done in an attempt to determine the accuracy of Meltzer and Lyon's physiological contentions have served to cast doubt upon them and upon the method derived from them. In the first place, Meltzer's original theorem of a contrary action has been pretty thoroughly discredited by the finding that a number of substances besides magnesium sulphate will produce the typical flow when they are injected into the duodenum. As most of these drugs do not relax the sphincter, it cannot be argued that they will reflexly stimulate the gall bladder. Furthermore, it cannot be shown on either animals or men that the flow of bile produced by magnesium sulphate is accompanied by a contraction of the gall bladder. It seems clear now that that organ is a weak and sluggish one which never contracts down like the urinary bladder does, even when it is stimulated directly by strong electric currents. When it does contract it produces pressures of at most 30 mm. of water, barely enough to overcome the resistance of the valves in the cystic duct. The writer has tried several times to stimulate the gall bladders of animals (anesthetized with urethane and opened under salt solution) into visible activity, but without success. We know also that at operations on men or on animals, the gall bladder is practically never found empty, as it would be if it occasionally squeezed out its contents. The available evidence suggests strongly that bile flows from the gall bladder, not because the muscle there has contracted, but simply because the pressure in the common duct has been lowered by the relaxation of the sphincter at the duodenal end.

Certainly, in the face of these physiological data, the users of the Meltzer-Lyon technic can no longer assume with any confidence that the dark bile comes from the gall bladder. There are a few experiments recorded which suggest that occasionally that may be the case, but most of them show conclusively that the color changes can be obtained in the entire absence of a gall bladder, that is, when that organ has been removed by operation or closed off by disease. It follows that if we cannot safely assume that the dark bile has come from the gall bladder we must not attempt to diagnose cholecystitis because we find bacteria or detritus in that bile. Furthermore, it must be remembered that although the duodenum contains fewer bacteria than does any other part of the bowel, it generally contains some; and it has been shown that an increase in their number will depend largely on the degree of acidity of the gastric juice, upon the amount of saliva swallowed, the sterility of the last food eaten, the condition of the teeth, etc. Another difficulty is that in a surprisingly large proportion of cases of gall bladder disease operated upon, no bacteria can be grown from the bile even with the most advanced cultural methods. Even in the presence of stones, more than 50 per cent of the biles are sterile. Unfortunately, few of the men who have tried to use the Meltzer-Lyon technic diagnostically seem to have been aware of these limitations and sources of error.

THERAPEUTIC PROBLEMS

It seems pretty clear from this review that as a diagnostic procedure the method has hardly a leg to stand upon. Let us see now if it fares any

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better when we come to examine its therapeutic pretensions. To begin with, we can admit two things: First, that it cannot be expected to have any influence on gall stones; second, that it may easily be of some benefit in the treatment of catarrhal jaundice. Even there, however, it is hard to say how much good it does because the patients get well anyhow, and one can never tell in a given case whether or not the period of illness has been shortened by the treatment. Thus, recently, one of my patients began to fade in about three days. If I had done as I had planned, and had given him one or two Meltzer-Lyon treatments, the outcome in his case would have furnished a strong argument in favor of the new technic.

The great difficulty in treating chronic cholecystitis is that we are faced with an infectious process which is localized not only in the walls of the gall bladder but, as Graham and Judd have shown, is generally scattered through the liver, through the glands about the cystic duct and often into the head of the pancreas. Even if we could clear up this infection with a few treatments, how could we hope to prevent its recurrence; and why should we expect a "non-surgical drainage" to succeed when the surgical drainage has so often failed? As is well known, a large proportion of the people who are relieved of their symptoms after operative drainage, return later for a cholecystectomy, because the infection has returned and new stones have been formed. Meyer, at the Hooper Foundation, has produced chronic recurring cholecystitis in animals simply by putting small foreign bodies into their gall bladders. This little handicap so lowers the resistance of the organ that although it may clear itself of the infection from time to time, it cannot remain clear. Similarly, a woman with gall stones or a scarred gall bladder, is subject to a flareup of infection whenever she has a cold, or whenever she puts undue strain on her digestive tract.

That being the case, it seems to me that there is only one thing to do with a chronically diseased gall bladder if we really want a cure, and that is to take it out. Individual cases will always have to be treated individually, however, and in the present state of our knowledge it is often hard to decide when a patient should be urged to go to the surgeon, and when she should be advised to let well enough alone.

I am glad to see that Lyon now admits that we must be careful how we speak of "cures" in an essentially chronic disease, and that he advises immediate operation in many of the bad cases. I agree with him that his technic is worth trying on certain people who for one reason or another cannot submit to operation, or who have had unsuccessful or partly successful operations. Furthermore, I think it should be tried in every rebellious case of catarrhal jaundice. The method may occasionally be helpful in establishing the presence of severe cholecystitis, but I would never think of relying upon it for the diagnosis of early or obscure forms of the disease.

LETHARGIC ENCEPHALITIS

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Epidemic Encephalitis began in Europe in 1916 and 1917, following in the wake of the influenza. Early the next winter the disease appeared in epidemic form in Paris. It then spread to England, where it was mistaken for Botulism, but the error was soon corrected by the investigation of McNalty, James and Marinesco and McIntosh. Encephalitis did not remain in Europe, but soon spread to Africa, Asia, Australasia and America, appearing here in the winter of 1918 and 1919.

It is interesting to note the spread of Encephalitis in America during the first six months of 1921. In New York there were 153 cases; in Connecticut 52; in Illinois 168; in Wisconsin 16; in Oregon 11; in California 30. The southeastern, southern and southwestern states seemed to have escaped, as only a few scattering cases were reported. The disease first made its appearance in Napa County the latter part of September and continued until the middle of October, claiming in Napa alone five patients, all of whom died. The disease met with at Napa was of a very virulent and rapid form, being of three distinct types: lethargic encephalitis, poliencephalitis and a milder type of encephalitis with a crisis and a rather rapid convalescence.

Age and Sex—There were five cases in all, the youngest patient being five years old and the oldest eighteen; two males and three females.

Relation to Influenza—This relation was direct; in all cases there was a history of influenza.

Mode of Onset—The premonitory symptoms were lassitude, general malaise, marked constipation, insomnia, pain in neck, jerking and twitching being frequent symptoms, with headache and gastro-intestinal disturbances culminating in vomiting.

Fever—All patients had fever, the temperature rapidly rising from normal to 105, with a slight drop before death.

Pulse—The pulse presented the usual features of infectious diseases, ranging from 100 to 120. At times the pulse was very weak and stimulation had to be resorted to.

Central Nervous System—Restlessness and insomnia were marked in all cases, especially the lethargic type which later developed listlessness, stupor, and a mask-like face. There were also present ptosis, diplopia, facial palsy, and partial opisthotonos with a characteristic pill-rolling posture of the hands. The patients remained in the state of lethargy and somnolence with a gradual darkening of the sensorium until pulmonary oedema caused death.

Cranial Nerves—Involvement of the third cranial nerve was shown by diplopia, also by ptosis which was present in three out of five cases. Involvement of fourth cranial nerve was shown by paralysis in three cases. Involvement of fifth cranial nerve was shown by difficulty in mastication in four cases. Involvement of the seventh cranial nerve was shown by incomplete facial palsy